IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Application No.	10/748,450	Art Unit:	1644
Applicant:	Richard L. Boyd	Examiner:	Michail A. Belyavskiy
Date Filed:	December 30, 2003	Conf. No.	2366
Docket No.	286336.150US1/NOR-011CP2	Cust. No.	23483
Title:	**Stimulation of Thymus for Vaccination Development**		

CERTIFICATION UNDER 37 C.F.R. § 1.8

I hereby certify that this correspondence is being deposited with the United States Postal Service as First Class Mail on the date indicated below and is addressed to: **Mail Stop Amendment**, Commissioner for Patents, P.O. Box 1450, Alexandria, VA 22313-1450.

10/10/2006
Date of Deposit

Rochelle Capobianco
Rochelle Capobianco

Mail Stop Amendment
Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

DECLARATION OF DR. RICHARD L. BOYD UNDER 37 C.F.R. § 1.132

Dear Sir:

In connection with the above-referenced patent application, I, Richard L. Boyd, declare as follows:

1. I received my Ph.D. degree in Immunology from Monash University, Australia in 1976. I was a Senior Tutor in the Department of Pathology and Immunology at Monash University from 1976-1977. I held a research fellowship at the University of Innsbruck, Austria, from 1978-1982, following which I held a Research Fellow position from 1983-1984. I was a Lecturer at Monash University from 1985-1994. Currently, I am an Associate Professor in the Department of Immunology at Monash University. I am also the Director of the Immunology Platform Program at the Australian Stem Cell Center and the Deputy Director of Monash Immunology

and Stem Cell Laboratories. I have authored or co-authored more than 193 journal articles in the area of immunology and presented over 500 oral presentations at conferences and research institutes. I was the Editor-in-Chief of *Developmental Immunology* from 1999-2003, and a member of the editorial boards of *Developmental and Comparative Immunology* (1993-2003) and of *Clinical and Developmental Immunology* (2004-2006). I review articles for numerous journals including, *Nature, Nature Immunology, Blood, Autoimmunity,* and the *Journal of Experimental Medicine.* I was the recipient of two awards from the Australian Federal Government and a co-recipient of two international prizes for my research. My *curriculum vitae,* which includes a list of my publications and presentations, is provided as **Attachment A** following ***page 8*** of this Declaration.

2. I am the Chief Scientific Officer of the Assignee of the above application

3. I am the sole inventor of the above-referenced patent application, and accordingly, have read and am familiar with the above-referenced patent application. I am also familiar with the Office Action dated April 10, 2006 (hereinafter "Office Action") in the above-referenced application.

4. As I understand the Office Action, the Examiner rejected the pending claims, in part, as not being enabling (*see,* Office Action, pages 4-7). The Office Action states, in relevant part, that:

> Since there is no animal model data and study in the specification showing the effectively [*sic*] for improving an immune response to a vaccine antigen by disrupting sex steroid mediated signaling through administering one or more pharmaceutical [*sic*] as claimed in claims 15, 30-33, 35, 36, 39-60, 63-68, 75 and 76, it is unpredictable how to correlate *in vivo* results of regeneration of thymus by removing the effects of sex steroid on the thymus after surgical castration with claimed method for improving an immune response to a vaccine antigen by disrupting sex steroid mediated signaling through administering one or more pharmaceutical [*sic*] as claimed in claims 15, 30-33, 35, 36, 39-60, 63-68, 75 and 76.
>
> In view of the absence of a specific and detailed description in Applicant's specification of how to effectively use said pharmaceutical composition and vaccine as claimed, and absence of working examples providing evidence which

> is reasonably predictive that the claimed pharmaceutical composition and vaccine are effective for in vivo use, and the lack of predictability in the art at the time the invention was made, an undue amount of experimentation would be required to practice the claimed method with a reasonable expectation of success.

5. I respectfully disagree with the Examiner's position. My invention is directed, in part, to a method for improving an immune response to a vaccine antigen in a patient. The method includes reactivating the thymus of the patient and administering a vaccine comprising a vaccine antigen to the patient. This method allows the patient to develop an improved immune response to the vaccine antigen. My patent application teaches a trained clinical immunologist how to make and use the invention as claimed in this application.

6. As a preliminary matter, with respect to the Examiner's comments in the paragraph bridging pages 6-7 of the Office Action, I note that my claimed invention is not directed to developing new vaccines but, rather, to enhance the application of marginally-effective vaccines or enhance efficacy in populations with impaired T cell responses. Such populations include patients who have undergone bone marrow transplant, chemotherapy and/or radiotherapy, and the elderly (Hertogh-Huijbregts *et al., Mech. Ageing Dev.* **53**:141–155 (1990); Nicoletti, *Immunobiology* **190**:127–137, 1994). The impaired T cell responses in these patients may be caused by: (i) lack of T cells generally, (ii) inappropriate T cell make up, and/or (iii) poor T cell response, among others.

7. It is well known that defective thymic function is very tightly linked to a defective immune system, and it is also widely accepted in the field that improved T cell number and function boosts immune responses (*see, e.g.*, Small *et al., Blood* **93**(2):467(1999), Mackall *et al., New Eng. J. Med.* **332**:143-149 (1995)), provided the improvement does not skew the TCR repertoire or produce inappropriate activation. A clinical immunologist would be well aware that an effective immune response to a vaccine requires functioning T cells. T helper cells (T_H cells) are required to initiate immune responses including the stimulation of B cells to produce antibodies and cytotoxic T cells (T_C cells) to produce cell-mediated cytotoxicity (*i.e.*, direct killing of cells

e.g. viral infected target cells or cancer cells) (Janeway *et al.*, Immunobiology: The Immune System In Health And Disease, 6th edition, Garland (2005), pp. 33, 361).

8. The anti-CD3/anti-CD28 model of T cell stimulation is widely accepted as an effective model in evaluating overall T cell response to antigen (*see, e.g.*, Abbas & Lichtman, Cellular and Molecular Immunology, 5th edition (updated), Elsevier Saunders (2005), p. 169; Verhoef *et al.*, *Scand. J Immunol.* **50**(4):427-432 (1999)). In **Exhibit A,** I provide the results of experiments showing responsiveness of murine T cells to T cell receptor (TCR) stimulation following sex steroid signaling disruption by castration. As you can see, responsiveness of T cells was significantly increased in 18-month old castrated mice compared with uncastrated young mice (2 months) and uncastrated older mice (18 months). Thus, sex steroid inhibition increased T cell responsiveness in aged mice.

In **Exhibit B**, I provide T cell stimulation data from a human study involving 42 patients (greater than 18 years of age and of both sexes), who were diagnosed with malignant or non-malignant haematological disorders requiring chemotherapy and allogeneic transplantation. The treated group (20 patients) received the LHRH agonist, Zoladex, for 4 months at doses of 3.75 mg/kg body wt/month for females, and 7.5 mg/kg body weight/month for males, which are the approved doses in Australia for endometriosis and prostate cancer, respectively. Blood was drawn prior to transplant and at intervals over the 12 months post-transplantation and stimulated with anti-CD3 (5 μg/ml) and anti-CD28 (10 μg/ml) antibodies. As you can see, the Zoladex-treated group showed statistically significant increases in T cell responsiveness *in vitro* to T cell stimulation through the TCR with anti-CD3/CD28 (*see*, **Exhibit B**). Thus, sex steroid inhibition increases T cell responsiveness of human T cells.

9. I would also like to direct the Examiner's attention to Figures 14 to 19 of my application, where mice were castrated and then inoculated in the hind foot-hock with 4×10^5 pfu of Herpes Simplex virus-1. Lymph nodes were analyzed 5 days post-inoculation for a primary immune response. This work serves as a model for response to vaccine, even though the mice were not

subsequently challenged with a further infectious dose. The loss of sex steroids improves T cell responsiveness and would be expected to improve resistance to challenge. An increase in the number of T cells able to react to antigen would be expected to result in an increase in the number of memory T cells available to respond to challenge. Although there was no follow-up challenge, the model provides an accepted means for assessment of the immune response to antigen challenge. The model is known to involve all aspects of an immune response, mimicking an injected vaccine, namely, the capture of antigen by antigen presenting cells (APC) at the site of entry (presumably by dendritic cells or Langerhans cells), its passage with the APC to the draining lymph node, the recruitment into the lymph node of potential target lymphocytes, the activation of those lymphocytes specific to the antigen and release back into the circulation of those lymphocytes not specific for the antigen (Janeway, pp. 417-426). This leads to development of effector T cells able to remove the virus (Janeway, p. 425). The model thus represents a general physiological immune response as produced by an effective vaccine. Figures 14b and 17b demonstrate that the mice with disrupted sex steroid signaling had a larger number of activated CD8 cells in the lymph node, indicating a greater response to the HSV inoculation, and Figures 15 and 19 indicate that these activated cells demonstrate the appropriate Vβ type. Figure 18 demonstrates that the inoculation produced a superior CTL response in old castrated mice compared to sham-castrated old mice. In summary, this disclosure, which is in the application-as-filed, would lead a clinical immunologist to recognize that sex steroid inhibition can improve a vaccine response.

10. Data presented in **Exhibit C** pertain to the immune response in aged castrated mice as a proof of concept for the well-known loss of immune capacity in aged humans and, hence, their decreased capacity for responding to vaccines. Mice were immunized intranasally with the Hkx31 influenza virus and culled 7 days later. The data show that when 15-month old mice were castrated 6 weeks prior to immunization, there was a reduction in the proportion of CD8+ T cells (**Exhibit C, Fig. a**) due to larger increases in other lymphoid cells *e.g.*, B cells and CD4+ T cells (data not shown), but importantly there was a statistically significant increase in the

number of CD8+ T cells (**Exhibit C, Fig. e**). T cell response to peptides (NP, PA, and PB) was poor as measured by cytokines necessary for protection against this virus (IFN-γ, TNF-α, and IL-2) (**Exhibit C, Figures f-h).** This is similar to the aging human population, which responds poorly to flu vaccines (Deng *et al., J. Immunol.* **172**: 3437–3446 (2004)). Defective CD8+ T cell expansion and low Th1 cytokines are linked to weak immune responses. As a corollary - reversing these would be expected to improve the immune response. When the mice were castrated, there was a significant increase in the production of these cytokines. **Exhibit C, Figure b** shows the proportion of T cells producing IFN-γ increased (reflecting the increase in reactive T cells), as did the total cell number of IFN-γ-producing CD8+ T cells (**Exhibit C, Fig. f**). These cells did not change in the proportion of cells co-producing TNF-α (**Exhibit C, Fig. c**) and IL-2 (**Exhibit C, Fig. d**), indicating that the immune response is normal in the types of cells being activated. These cytokines are widely recognized as being the essential mediators of immunity to viral infections - particularly those requiring Th1 cells (Janeway, pp. 347, 361), which are generally considered the appropriate response for flu. For a vaccine to be successful, adequate levels of these cytokines need to be generated; this is now occurring as a result of the reduction in sex steroids. The consequences of antigen recognition by an armed effector T cell are determined largely by the set of effector molecules it produces on binding a specific target cell (Janeway, p. 351). Thus, the loss of sex steroids increased the number of cells responding to the flu peptides and, hence, improved the ability of the immune system to respond to a vaccine and defend against infection.

11. Finally, in **Exhibit D**, I provide data that show improved vaccine responses as claimed in this application. The data relates to the effect of sex steroid inhibition on the *Hemophilus influenza* B (HIB) vaccine response in human patients. A group of 17 allogeneic bone marrow transplant (BMT) patients were involved in this study: 11 patients received LHRH analog treatment, while 6 patients did not and served as the control group. The study was conducted as follows: (1) serum samples were collected from the patients 5 months after BMT (the pre-immunization bleed); (2) the patients were vaccinated at 6 months post-transplant with

PedvaxHib (*Hemophilus influenza* vaccine); and (3) a second serum sample was collected from the patients 9 months post-BMT (post-immunization bleed). The response to vaccination was measured using BINDAZYME™ Enzyme Immunoassay kits, (The Binding Site (UK)) with an assay sensitivity of +/- 0.096 μg/ml. Antibody responses above 1.0 μg/ml are considered to be protective (Mariani *et al., Clin. Diag. Lab. Immunol.* 667–674 (1998)).

None of the 6 patients of the untreated group showed response to vaccination. However, 3 of the 11 patients of the treated group showed response to vaccination as demonstrated by a more than a 2-fold increase in antibody level (*see*, **Exhibit D**). Those 3 patients had ~6.3, 8.5, and 14.9 μg/ml of antibody difference between pre- and post-immunization, whereas the biggest difference observed in the control group was only 1.4 μg/ml. The overall antibody response was decreased in the control group from an average of 3.1 to an average of 2.5, whereas in the treated group the vaccine response increased from an average of 2.5 to 3.9 (*see*, Tables 1 and 2).

TABLE 1: CONTROLS (6)

PATIENT	SEX	AGE	ILLNESS	Pre-Immun. 5 MONTHS (μg/ml)	Post-Immun. 9 MONTHS (μg/ml)	DIFF. (μg/ml)
ASW	F	51	HODGKINS	**1.4**	1.9	0.5
DMH	M	34	AML	**2.8**	0.9	-1.9
LJS	F	43	CML	**1.4**	2.8	1.4
JM	M	48	HODGKINS	**3.1**	4.3	1.2
NLS	F	29	HODGKINS	**3.7**	0.5	-3.2
SJM	F	29	ALL	**5.9**	4.4	-1.5
AVERAGE				3.1	2.5	-0.6

TABLE 2: LHRH PATIENTS (11)

PATIENT	SEX	AGE	ILLNESS	Pre-Immun. 5 MONTHS (μg/ml)	Post-Immun. 9 MONTHS (μg/ml)	DIFF. (μg/ml)
AAH	M	55	AML	**1.1**	0.4	-0.7
BFH	M	53	AML	**2.7**	**17.6**	14.9
BNB	M	44	AML	**4.2**	**1.7**	-2.5
IJM	M	43	ALL	**2.5**	**8.8**	6.3
JET	F	36	AML	**2.3**	0.2	-2.1
JCM	M	43	Myelo-dysplasia	**2.0**	0.6	-1.4
KLB	F	43	AML	**1.0**	**9.5**	8.5
MRP	M	28	ALL	**2.4**	**1.2**	-1.2
PGM	M	35	AML	**5.1**	0.4	-4.7
TAH	M	23	AML	**1.9**	**1.9**	0.00
ZEH	M	35	NHL	**2.4**	**1.0**	-1.4
AVERAGE				2.5	3.9	1.4

12. In summary, based on the discussion above, I respectfully aver that the instant application does teach how to make and use the invention as claimed.

13. I further declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true and further that these statements are made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code and that such willful false statements may jeopardize the validity of the application or any patent issuing thereon.

Richard Boyd.

Date: October 10, 2006

Richard L. Boyd, Ph.D.

ATTACHMENT A

Attached is the *curriculum vitae* of Dr. Richard L. Boyd.

CURRICULUM VITAE

Richard Boyd

October 2005

Born: 18th November 1950, Melbourne, Australia
Marital Status: Married
Nationality: Australian

Academic Qualifications

1973-76 Doctor of Philosophy
Department of Pathology and Immunology
Monash University, Victoria, Australia
Thesis Title: Chicken Lymphoid Tissues: Cellular Differentiation, Microenvironment and Ontogeny

1972 Bachelor of Science, Honours (First Class)
Department of Pathology and Immunology,
Monash University
Thesis Title: Lymphocyte Differentiation - Membrane Antigenic Changes Associated with Blast Transformation

1969-71 Bachelor of Science, Monash University, Australia
Majors: Biochemistry, Genetics

Academic Appointments

Current Appointments

2005- Deputy Director, Monash Immunology and Stem Cell Laboratories, Monash University

2003- Director, Immunology Platform Program, Australian Stem Cell Centre

1995 - Reader / Associate Professor, Department of Immunology (formerly Pathology and Immunology), Monash University.

Previous Appointments.

1989 – 94 Senior Lecturer (tenured appointment)
Department of Pathology and Immunology, Monash University.

1988 Lecturer (tenured appointment)
Department of Pathology and Immunology, Monash University

1985 – 87 Lecturer (fixed term)
Department of Pathology and Immunology, Monash Univeristy

1984 Research Fellow II,
Department of Pathology and Immunology, Monash University

1983 Research Fellow I
Department of Pathology and Immunology, Monash University.

1980-82 Research Fellow, Tenured position
(Universitats Vertragsassistent), University of Innsbruck, Austria.

1978-79 Research Fellow,
(Universitats Vertragsassistent), University of Innsbruck, Austria.

1976-77 Senior Tutor
Department of Pathology and Immunology, Monash University.

Fellowships

1983 -84 Monash University Post-doctoral Research Fellowship.

1973 -76 Commonwealth Postgraduate Research Award.

1969 -72 Victorian Education Department Studentship.

1967 -68 Victorian Education Department Bursary.

1991 Division of Rheumatology/Allergy, Clinical Immunology, University of California, Davis, California. March - April, 1991. Guest Faculty Professor, University of California.

1992 Institute for General and Experimental Pathology, University of Innsbruck, Innsbruck, Austria. April 1992 - September 1992. Guest Professor of the Austrian Academy of Sciences.

1992. Institute for Virology and Immunobiology, University of Wurzburg, Wurzburg, Germany. Guest Professor of the German Research Foundation, Special Research Division (Deutsche Vorschungsfond, Sonderforschung Bereichshaft). September December, 1992.

1992 –93 Eleanor Roosevelt Fellowship, UICC American Cancer Society International Cancer Research Fellowship. (International Union Against Cancer).

1993 Department of Industry, Technology and Regional Development.

(Fellowship for the Ludwig Institute for Cancer Research, Lausanne, Switzerland).

1994 Institute for General and Experimental Pathology, University of Innsbruck, Innsbruck, Austria. February. Guest Professor of the Austrian Academy of Sciences.

1995 Department of Industry, Technology and Regional Development (Fellowship for visit to the Department of Immunology, Masachussetts Institute for Technology, Cambridge, Masachussetts)

1996 Department of Industry, Technology and Regional Development (Fellowship for visit to Ontario Cancer Research Institute, Toronto, Canada).

1999 Institute for General and Experimental Pathology, University of Innsbruck, Innsbruck, Austria. January. Guest Professor of the Austrian Academy of Sciences.

Awards

2003 Australian Federal Government Centenary Medal for Service to International Medical Research and Undergraduate Teaching

2004 Australian Federal Government Business/Higher Education Round Table Award for Outstanding Achievement in Research and Development and Education and training.

Prizes

1981 Preis der Stiftung der Hoechst Aktiengesellschaft, 1981
Effector mechanisms in spontaneous autoimmune thyroiditis in obese strain chickens. R.L. Boyd and G. Wick

1982 Von Basedow-Forschungspreis, 1982
Effector mechanisms in the spontaneous autoimmune thyroiditis of obese strain chickens : analysis of cytotoxic cells.
R.L. Boyd and G. Wick.

Involvement in Current University Initiatives

Executive meetings for Monash Immunology and Stem cell Laboratories, Department of Immunology, and School of Biological Science executives; Animal Ethics.
In addition the following more specialised responsibilities:
1. Member, Forward Planning Committee, Monash University Biomedical Institute (2005 -)
2. Member, Faculty of Medicine Delegation to China, October 27, 28, 2005.
3. Member, Monash-Newcastle (UK) committee for Collaborative Initiatives in Ageing and Stem Cell research (Prato meeting, Italy) (2005 -).
4. Member, committee for collaboration between Monash University and the University of California, San Diego, and University of California, Davis –to establish joint laboratories in stem cells and regenerative medicine. (site visit July 2005)

Society Memberships
Australian Society for Immunology

IgV – Victorian Branch of Australian Society for Immunology (Foundation Committee member, 1995)
International Union Against Cancer (UICC). Life Member
International Society for Stem Cell Research (ISSCR)

Editorial and Scientific Appraisal Services
Editor-in-Chief Developmental Immunology 1999 - 2003
Member Editorial Board, Developmental and Comparative Immunology. 1993 – 2003
Member Editorial Board, Clinical and Developmental Immunology 2004-

Review for:
Autoimmunity
Blood
Experimental Biology and Medicine
European Journal of Immunology
Immunity
Immunology and Cell Biology
Journal of Autoimmunity
Journal Clinical Investigation
Journal Experimental Medicine
Journal of Immunology
Nature
Nature Immunology
Nature Reviews Immunology
Proceedings National Academy of Sciences
Trends in Immunology

Review Grants and Fellowships for:
Alfred Hospital, Melbourne
Anti-Cancer Councils of Victoria, New South Wales and Queensland
Australian Research Council
Human Frontier Science Program
MRC United Kingdom
National Institutes for Health, Bethesda
NH&MRC, Australia
New Zealand HRC
Raine Foundation, Western Australia
USA - Israel Binational Science Foundation

International Committees.

1. Member, Organising Committee, German Society for Immunology, Innsbruck, 1979.

2. Member, Cluster of Thymic Epithelial Staining (CTES) nomenclature committee (1989 - 1995).

3. Member, Organising Committee for Australian Society for Immunology Annual Scientific Meeting, Melbourne, 1990.

4. Member, Avian Leucocyte CD nomenclature committee (1990 - 2000).

5. Member, Scientific and Organising Committees, Foundation member, International Workshop on the Thymus. Rolduc, The Netherlands.1988, 1991, 1995, 1997, 2001, 2004.

6. Chairman, Organising and Scientific Committee, International Workshop on T Lymphocytes, Heron Island. ThymOz I Oct 11 -15, 1995, ThymOz II March 25 - 29, 1998, ThymOz III April 11-18, 2000, ThymOz IV April 1-5, 2003, ThymOv V April 5-10, 2005

7. Co-chairman, Organising Committee for International Conference on Germinal Centres and Lymphatic Tissues in Immune Reactions, Graz, Austria. 1996.

8. Co-Chairman, Organising Committee, Australian Society for Immunology Annual General Meeting, Melbourne, Dec 1998.

9. Member, Scientific Committee, 14th International conference on lymphatic tissues and germinal centres in immune reactions. Groningen, June 23-27, 2002.

10. Member Scientific Organising committee, 2nd Australian Stem Cell Conference, Sydney. 2004.

11. Member, Scientific Committee, 15th International conference on lymphatic tissues and germinal centres in immune reactions. Potsdarm. Germany. 2005.

12. Chairman, organising Committee, "Rebuilding Immunity for Better Health – an interface between immunology, stem cells and regenerative medicine." Monash University, October 6 – 7th, 2005.

Invitations to Conferences

More than 500 oral presentations to conferences and research institutes.

1. 1984 I.U.I.S. Symposium on Autoimmunity, March 18-24 Igls, Austria.

2. 1998 Invited Speaker, Spring Session, Tasmanian Haematology, Immunology and Neoplasia Group, Ross, Tasmania. April.

3. 1988 Invited Speaker, The Thymus: Histophysiology and Diagnosis in the Immune System. Rolduc. The Netherlands. May.

4. 1988 Invited Speaker, Understanding Modern Biotechnology Workshop, Monash University. July.

5. 1990 Invited Chairperson, Thymus Workshop: Conference on T lymphocyte Development and function, Rolduc, The Netherlands. May.

6. 1990 Invited Symposium Speaker, Annual Meeting, Australian Society for Immunology, Melbourne. December.

7. 1991 Invited chairperson, 10th International Conference on Lymphatic Tissues and Germinal Centres in Immune Reactions, Compiegne, France. July.

8. 1992 Invited Chairperson and Speaker, 7th. Winter Conference on Controversial Immunology, Igls, Austria. April.

9. 1992 Invited Chairperson, Thymus Workshop: Conference on T lymphocyte Development and function, Rolduc, The Netherlands. May.

10. 1992 Invited Symposium Speaker, Annual Meeting Australian Society for Immunology, Auckland. December.

11. 1993 Invited chairperson, 11th International Conference on Germinal Centres and Lymphatic Tissues in Immune Reactions, Liege, Belgium. June.

12. 1993 Invited Symposium Speaker, Annual Meeting of the Austrian Society for Allergy and Immunology, Graz, Austria. July.

13. 1993 Invited chairperson and Symposium Speaker, Joint Meeting, Australasian Society for Immunology / Society for Leucocyte Biology, Sydney. December.

14. 1994 Invited Symposium Summary, Thymus Workshop: Conference on T lymphocyte Development and function, Rolduc, The Netherlands. May.

15. 1994 Invited Symposium Speaker, International Conference on Scleroderma, Sydney. July.

16. 1994 Invited speaker, Immunology and Developmental Biology of the Chicken, Basel Institute for Immunology, Basel, Switzerland. November.

17. 1995 Invited Co-chairman, 9th. International Congress of Immunology, San Fransisco, California. USA. July.

18. 1996 Invited Symposium Speaker, Australian Poultry Science Symposium, Sydney, Feb 6-7.

19. 1996 Invited Symposium Speaker, International Symposium on Avian Endocrinology, Alberta, Canada, March 31 - April 5.

20. 1996 Invited Keynote Speaker, Avian Immunology Workshop, Obergurgl, Austria, April 21 –24.

21. 1996 Invited Symposium Speaker, 5th International Expert Forum on Immunotherapy and Gene Therapy, Jerusalem, June 4-9, 1996.

22. 1996 Invited Symposium Speaker and Workshop Chairman, 12th International Conference on Lymphoid Tissues and Germinal Centres in Immune Reactions, Graz, Austria, July 1-5, 1996.

23. 1996 Invited Plenary Session Speaker, First Congress of the Federation of Immunological Societies of Asia-Oceania, (ASI) Adelaide, Dec 1-5.

24. 1997 Invited Chairperson 5th Conference of the Immunology Group of Victoria, Mt. Buffalo, March 16-18.

25. 1997 Invited speaker, International Workshop on Thymus, Rolduc/Kerkrade Holland, March 23-26.

26. 1997 Invited Symposium speaker 5th Kyoto T Cell Meeting, Kyoto, October 2-4.

27. 1997 Invited Symposium speaker and Chairperson, XIIIth Int Congress of Comparative.Endocrinology, Yokoma Nov 17-21.

28. 1998 Plenary Chairperson, symposium speaker, 25th Annual Meeting Australian Soc Immunology, Melbourne Nov 29-Dec3.

29. 1999 Invited Speaker, FASEB/Clinical Immunology. Washington April 19-21.

30. 1999 Invited Speaker, Rolduc International Workshop on Thymus, May 1-4.

31. 1999 Invited Speaker, Austrian Academy of Science Special Symposium in Honour of G. Wick, Innsbruck, May 8.

32. 1999 Invited Symposium Speaker, 6th Asia/Oceania Regional Congress of Gerontology, June 8-11.

33. 1999 Invited Speaker and Workshop Chairman, 13th International Conference on Lymphoid Tissues and Germinal Centres in Immune Reactions, Geneva, Switzerland 1-6.

34. 2000 Invited Symposium speaker and Chairperson, VII International Society Avian Endocrinology, Varanasi, India, Feb 1-5.

35. 2000 Invited Symposium speaker, XXI World Poultry Congress, Montreal, August 20-24.

36. 2000 Invited Chairperson. Australian Society for Immunology, Sydney. Dec.

37. 2001 Invited Symposium speaker. Thymus Workshop. Rolduc. Kerkrade. The Netherlands, May 12-15.2001.

38. 2001 Invited Speaker: IgV Meeting, Mt Buffalo, Victoria, October 14-16.

39. 2001 Invited Keynote Address, ThymUS International thymus meeting, Peurto Rico, November 2-6.

40. 2001 Invited Chairperson, ThymUS International Thymus Meeting, Peurto Rico, November 2-6.

41. 2001 Invited Chairperson, Australian Society for Immunology, Canberra. December 1-4.

42. 2002 Invited symposium speaker, Kyoto T Cell Conf. (KTCC), Kyoto, Japan, April 3-5.

43. 2002 Invited Symposium Speaker and Chairman, 14th International Conference on Lymphoid Tissues and Germinal Centres in Immune Reactions, Groningen, The Netherlands, June 23-27.

44. 2002 Invited Symposium Speaker, Federation of Immunological Societies of Australasia, Beijing China, October.

45. 2002 Invited key Note Speaker, Australian Society of Medicine, Canberra, Nov 21-23,

46. 2003 Invited speaker and corporate presentation Bio 2003, Washington June 22-27.

47. 2003 Invited Speaker, Workshop chairman, 1st National Stem cell Conference, Melbourne Oct 9-11.

48. 2003 Invited Speaker, First Barossa Meeting (Science amongst the Vines), "Regeneration". Barossa Valley South Australia. Nov 19-21.

49. 2003 Invited Symposium Speaker, Japanese Society for Immunology, December, 7-10.

50. 2004 Invited Symposium speaker. Swiss Society for Allergy and Immunology, Geneva April 15-17.

51. 2004 Invited Speaker, Rolduc International T cell Workshop, Rolduc. The Netherlands. May 1-5.

52. 2004 Invited speaker, Japanese Society for the Promotion of Science, Core-to-Core Workshop on Thymus Organogenesis' . Tokushima August 16-18.

53. 2004 Invited Speaker, ThymUs - International Workshop on the Thymus. Peurto Rico, November.

54. 2004 Invited Speaker and Symposium Chairperson, 2nd Australian Stem Cell Conference/ Australian Health and medical Research Congress, November, Sydney.

55. 2004 Invited Speaker, International Conference on NKT cells, Heron Island, 8th -13th September

56. 2004 Invited Symposium Speaker, British Society for Immunology, December 10th, Harrogate. UK.

57. 2004 Invited Symposium Speaker, Australian Society for Immunology, Adelaide, December

58. 2005 Invited Speaker, Lorne Cancer Conference, Phillip Island, Victoria, February

59. 2005 Invited Speaker, 8th International Symposium on GnRH Analogues in Cancer and Human Reproduction, February, Salzburg, Austria.

60. 2005 Invited Speaker, Kyoto T Cell Conference, April 6-10, Kyoto, Japan

61. 2005 Invited Speaker, Japanese Society for the Promotion of Science, Core-to-Core Workshop on Thymus Organogenesis' April 11-12. Tokushima, Japan.

62. 2005 Invited Speaker, 3rd Congress of Federation of Immunology Societies of Asia-Oceania (FIMSA), Hangzhou, April 18-22.

63. 2005 Invited Speaker and Scientific Committee member, 15th International conference on lymphatic tissues and Germinal Centres in Immune Reactions . April 20-24 Potsdam, Germany

64. 2005 Invited Speaker Advances in Agricultural and Medical Biotechnology, 29-30 September Kuala Lumpur.

65. 2005 Invited Speaker, 6th Australian Peptide Conference, October 9-14, Hamilton Island, Queensland.

66. 2005 Invited Speaker, 10th Annual Australian Autoimmunity, 19-21st October.

67. 2005 Invited Speaker, Science in the Vines, November, Barossa Valley. South Australia

68. 2005 Invited Symposium Speaker. 35th Annual Meeting Australasian Society for Immunology. December 4-8 Melbourne.

69. 2005 Invited Lecturer, Post-graduate Teaching Workshop, 35th Annual Meeting Australasian Society for Immunology. December 4-8 Melbourne.

70. 2005 Invited Speaker, Joint meeting of Monash and University of Newcastle, Prato, Italy February 27 – March 1.

71. 2006 Invited Speaker, Discovery Science and Biotechnology, Melbourne May 5-8.

Postgraduate Student Supervision

PhD Completed	27
PhD Current	14
M.Sc. Completed	2
M.Sc. Current	1
B.Sc. (Hons) completed	61
B.Sc. (Hons) current	2

Community Service

Member, Committee Sandringham Little Athletics Club: 1992 – 2001

Team Manager, East Sandringham Junior Football Club: 1996 – 2001

Member Sandringham Primary School Council: 1999 – 2002

Member Premises Committee, Sandringham Primary School: 1997 – 2001

Member Sandringham Primary School committee – Looking to Sandy's Future: 2000

President Sandringham Little Athletics Club: 1996 – 2001

Life Member, Sandringham Little Athletics Club 2001-

Publications

1. **Boyd, R.L.**, Rolland, J.M. and Cauchi, M.N. (1974) Membrane antigenic changes associated with PHA transformation of mouse spleen cells *in vitro*. Immunolog. Commun., **3** : 337-349.

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12. **Boyd, R**. and Wick G. (1979) Role of suppressor cells in autoimmune diseases. In European Surgical Research. Ed. W. Brendl, (S. Karger Verlag, Basel) pp.212-213.

13. **Boyd, R**. and Ward H.A. (1979) Antigenic changes associated with chicken B lymphocyte differentiation. Immunobiol. **156**:186.

14. **Boyd, R.**, Schauenstein, K. and Wick, G. (1979) Characterization of effector cells in spontaneous autoimmune thyroiditis. Immunobiol. **156**:244.

15. **Boyd, R.L.** and Wick, G. (1980) Killer cells in the chicken : a microcytotoxicity assay using antigen-coated erythrocytes as targets. J.Immunol. Methods. **35**: 233-247.

16. Wick, G. and **Boyd, R.** (1980) Effector and suppressor cells in Obese strain (OS) chickens with spontaneous autoimmune thyroiditis. Fed. Proc. **39**: 570.

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